THE BRITISH VETERINARY JOURNAL

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Vol. 107 **April, 1951** **No. 4**

EDITORIAL

VETERINARY PATHOLOGY

PATHOLOGY is a term that is continuously used to include many aspects of disease, their causes, preventions and cures. We are apt to forget that this general term may cause us to give too little attention to the part of this vast subject which deals with the processes of disease, viz., morbid anatomy and histology. There was a time in the history of veterinary science when much stress was laid on morbid anatomy and histology: we have only to refer to the writings of many workers on disease problems in the early days of research to show how much time was given to the detailed examinations, especially microscopical, to show how important this subject was considered. The tendency seems to have been to devote less and less time and study to this aspect of disease and to concentrate more upon cause than effect. There are still, of course, those who make a practice of including in their work detailed examinations of the various tissues and structures, from which much valuable information can be derived. On the whole, however, the tendency seems to be to neglect in some measure this important work.

There is to-day a marked scarcity of veterinary personnel well trained in pathological techniques and efforts are now being made to encourage young graduates to undergo the necessary training and to become highly proficient in this branch of veterinary science. As time goes on, the indications are that there will be a group of veterinary pathologists who will take their place in the ranks of pathologists generally and devote their lives to the detailed study of disease processes occurring in animals and link with pathologists in human medicine, comparing diseases in human beings with those affecting the lower animals. During the past few years we have seen greater interest being taken in this

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subject and also the efforts which some young veterinary graduates are making to obtain the necessary efficiency in the technical procedures and the interpretation of the results.

Again, although the tendency has been to neglect the subject to some extent, we have never been entirely without our veterinary pathologists in this country. They have done noble work and have shown us very clearly the need for this type of work and study. They have also continued to assert the need for more and more detailed study of disease processes in the elucidation of some of the difficulties in the understanding of disease in the animals with which we have to deal. Their efforts have gone far to clear up some of the true causes of apparently obscure conditions and, by their association with pathologists in human medicine, have demonstrated the similarities and differences in the disease conditions in the two branches of medicine. We look forward to the time when there will be no cause to criticise the position of true pathologists in veterinary medicine nor to have to draw comparisons between the time and amount of work devoted to the subject in the study of diseases of the human subject and those affecting animals.

In this issue of THE BRITISH VETERINARY JOURNAL there is published an article which illustrates the necessity for and the valuable results of detailed examinations of tissues and structures in different animals, leading to a better appreciation of the likely causes of the abnormalities. In his general comments the author expresses the opinion that questions of economics may have some influence on the amount of time and study given to our work on animal diseases. Undoubtedly, this may have an important bearing on our outlook, but it must not be our excuse for failing to adopt procedures which will advance our general knowledge and enable us to take our rightful place in the study of comparative pathology.

GENERAL ARTICLES

MYOPATHIES IN ANIMALS

A record of some cases including progressive muscular dystrophy (pseudo-hypertrophic) (dog), "weisses Fleisch" (lamb), neuropathic muscular atrophy (sheep) and lymphocytic/histiocytic myositis, neuritis, radiculitis (dog)

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Introduction

THE myopathies occupy a niche in human pathology and little is known about their origin and cause. The comparable position in veterinary medicine is confusing, due to inadequacy of pathological knowledge and paucity of material studied. A few cases are recorded and illustrated, for—apart from rarity—they might be of interest to clinicians and comparative pathologists.

So that direct comparison may be made with human conditions, which is an essential consideration of any animal disease, and so that terminology may be properly used, a brief summary is given of knowledge as available from recognised text-books on neurology (Kinnier Wilson, 1940; and Russell Brain 1940) and a few selected original articles (Bramwell, 1925; and Nevin, 1936 and 1938); text-books on general pathology devote relatively little space to these disorders of skeletal muscle, and all recount a comparable story (cf. Beattie and Dickson, 1943; Boyd, 1944 and 1947; Bell, 1944; and Anderson, 1948). Certain muscular atrophies dependent on disease of the central nervous system (e.g., amyotrophic lateral sclerosis) stand apart from the group known as muscular dystrophies. The belief is losing ground that in the latter the diseases are of primary muscular origin; they may be essentially disturbances of function located in the fibres or myoneural junctions. They most commonly become manifest in childhood, often have a familial character, and males are more prone. The outstanding symptom is progressive muscular weakness, and affected muscles may show true hypertrophy, pseudo-hypertrophy or atrophy. The muscles mostly concerned are those involved in fixation: hence the *Mm. gastrocnemius, deltoideus, infraspinatus* and *supraspinatus* are frequently affected. Most references stress that the basic pathology is a common one but the changes may vary in different muscles; the fibres may be swollen and rounded, with loss of striation, and the sarcolemmal nuclei may be increased; the fibres may be shrunken and stages of degeneration ranging to complete loss may be found; with these alterations there is a concomitant replacement of muscle by fibrous tissue and fat, the latter in the pseudo-hypertrophic form producing the macroscopic enlargement; infiltrations by lymphocytes and multinucleated giant cells

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may be present. The nature is apparently puzzling; the diseases are often congenital, and while many cases are inherited the mode of inheritance is not understood. It is not known whether the fault lies in the structure of muscles, in their metabolism, in the sympathetic nervous system, in the motor end organs, or in the endocrine control. Sometimes there is an associated endocrine complex with hypophyseal changes (as in Fröhlich's syndrome), with adiposity, sexual infantilism and acromegaly. The term " progressive muscular dystrophy " thus includes sub-divisions based on clinical findings, age of onset, a consideration of heredity, and the group of muscles affected.

Discussions then proceed to embrace other diseases: (1) *Dystrophia myotonica*, an hereditary disorder characterised by dystrophy, myotonia, cataract, and gonadal atrophy; (2) *Myotonia congenita* (Thomsen's disease), a rare hereditary disease distinguished by prolonged tonic contraction and retarded relaxation of muscles in which there are no lesions in the muscles or nervous system; (3) *Myasthenia gravis*, a chronic disorder with abnormal fatiguability due to some interference in conduction in the myoneural junctions and conditioned by pathological changes elsewhere in the body; and (4) a group of muscular diseases associated with thyroid disturbance, the relationship to which is complex.

In veterinary literature there are no authenticated recorded conditions analogous with the muscular atrophies intervolved with specific hereditary, or degenerative, diseases of the central nervous system such as amyotrophic lateral sclerosis. A peculiar hereditary lethal for localised and preganglionic motor neurones with spinal muscular atrophy in dogs has been described by Stockhard (1936). The disease occurred in crosses of Great Danes with St. Bernards and Great Danes with Bloodhounds; both parent stocks contributed to the complex of genes underlying the paralysis in each case. The puppies became paralysed between eight and fourteen weeks after birth, and there was muscular atrophy of the limbs associated with loss of motor neurones in the spinal cord. This occurred in a large canine breeding station, and although infection was stated to be excluded some of the microphotographs of the lesions might be reminiscent of some virus infections; at the magnification used, however, it is difficult to assess the exact nature of the lesions. The atrophied muscles were apparently not examined histologically. There are also no clear parallels with the syndromes in man known as *Dystrophia myotonica*, *Myasthenia gravis*, or those forming a complex with thyroid disorders. An interesting, and important, condition of goats was investigated very fully by Kolb (1938), but has attracted no attention of veterinarians. (In general the goat seems to have been neglected as an object of study in pathology, as may be seen from the " Handbook on Goat Diseases " produced by the National Veterinary Medical Association, Great Britain [1950].) Kolb recounts that this disorder in goats was known in the U.S.A. since 1904 in a breed which, he says, was introduced into Texas to uphold the veracity of a Tennessee farmer! The origin of the breed is obscure; they were snow-white mixed with Angora, and at one time scaring them was an apparently popular pastime. When startled and run around,

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they became rigid in all four extremities and were unable to move for eighteen to thirty seconds, but this was not accompanied by loss of consciousness. The condition was hereditary, but the mode of inheritance was not unravelled. Goats did not develop any malady until some time after birth, but it was seen once by Kolb in a kid three hours old. It tended to be more severe in cold weather, but the symptoms disappeared during pregnancy and could not be induced again until one month after parturition nor in the kids until they were one month old. Typical myotonic reactions followed percussion of muscles, and biopsy examinations showed an absence of lesions; quinine and other cinchona derivatives relieved the myotonic states, while prostigmine and pilocarpine aggravated the symptoms. Kolb concluded that all of these facts betokened that this caprine disease was practically identical with *Dystrophia congenita* (Thomsen's disease) in man and was thus an invaluable object for experimental study by medical workers. Brown and Harvey (1939) subsequently made elaborate physiological studies on other affected goats and showed that the muscles were not abnormally sensitive to acetylcholine but were to potassium ions; they concluded that the condition was an abnormality in the muscle fibre and that the neuromuscular apparatus was not involved. It is undetermined whether this caprine myotonia can be collated with a disease of baby pigs which has occurred in the U.S.A. for many years and to which Kernkamp (1950) has again drawn attention. In the pigs, symptoms are referred to as repetitive clonic contractions of voluntary muscles, often of the whole body. This disease (seen personally but never investigated in England) requires more intensive study for, as far as I know, no reports of pathological examinations of the muscular and nervous systems, or of clinical observations such as those of Brown and Harvey in the caprine myotonia, have appeared.

In consideration of the muscular dystrophies in animals, Nieberle and Cohrs (1949) state that only the simple forms play any role; these are linked with senile decay, cachexia, inactivity or destruction of nervous control by lesions in the central nervous system (see also reference to Stockhard, 1936). Muscular wasting in animals due to injury or pressure on parts of the brain, cord or peripheral nerves are well known. They also deal with so-called *Atrophia lipomatosa* in cattle and pigs, in which the muscles are more voluminous, are partially replaced by fat, and in which degenerative changes may be present in the muscle fibres; they compare this with the pseudohypertrophic form of muscular dystrophy in man. This condition must be distinguished from a simple fatty degeneration of suckling lambs and pigs associated with some anæmia and which has been thought to be congenital; it is described in many German textbooks and some reference is made to it by Hutyra, Marek and Manninger (1946); it is not mentioned by Thornton (1949), but he describes a fatty replacement type of change in muscles of slaughtered cattle and pigs in Britain. Joest (1929) treats the whole subject in greater detail but in similar terms. Two cases of progressive muscular dystrophy in dogs are specifically described: in one no autopsy was made and the diagnosis was based on clinical grounds; the other showed systemic atrophy of the entire

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body musculature with fibrous and fatty tissue replacement, but with no changes in the nervous system. Attention is also given by Joest to *Atrophia pseudo-hypertrophica lipomatosa* under the general heading of muscular hypoplasia (see also case of László, 1938).

In this same type of German literature there is always reference to the condition known as "weisses Fleisch," "Fischfleisch" or "Hühnerfleisch" in slaughtered calves, pigs and sheep. The disease may affect single muscles, groups or even the whole body, and as the names indicate the involved parts (or streaks in one muscle) have the pallid colour of chicken, or fish, flesh. Sometimes the muscles are œdematous, but they may be dry. Histologically the fibres are thickened, striation is lost, and there are hyaline, waxy or fatty changes in the fibres; fibrillary separation ensues, the sarcolemmal nuclei may be increased, marked interstitial infiltrations of lymphocytes and histiocytes may be present, and calcifications may be outstanding in old lesions. The ætiology for a long time was very obscure, and opinions veered around ideas on infections, trauma during transport, and pigment anomalies, for myoglobinuria may occur.

Reference must be made to the original work of Hjärre and Lileengen (1936) on this waxy degeneration in calves; they traced the literature back to 1866 to indicate that this same condition has occurred widespread in most of the continental countries of Europe since that time. They describe in detail the histological changes, which are like those already given above, and they proved experimentally that the same lesions could be produced in calves (two to three months old) as a result of a vitamin C deficient diet. By so doing they rendered obsolete a concept that the bovine species does not suffer from any scorbutic process on the basis that they are known to be able to synthesise ascorbic acid. Hjärre's results indicate that age has some influence; apart from the muscular lesions typical changes were found in the bones and teeth of the experimental calves which are known to be diagnostic of classical scurvy of the monkey, guinea-pig and man. (An excellent account of "weisses Fleisch" is also given in the valuable monograph of Seifried [1943] on vitamin deficiencies of domesticated animals.) It is important to remember that nutritional muscular dystrophy was first produced many years ago in laboratory animals fed on diets deficient in vitamin E (tocopherols); all the appropriate references can be obtained from the books of Seifried (1943), Spillane (1947) and Follis (1948), and need not be considered here. The lesions were shown to be identical with those described originally by Goettsch and Pappenheimer (1931) (see Pappenheimer [1939]). The concept was thus engendered that the tocopherols might be concerned ætiologically, and thus used therapeutically, in muscular dystrophies and amyotrophic lateral sclerosis in man. According to Spillane (1947), the position is by no means clear and claims on the beneficial effects of wheat germ oil have been prematurely made (see Bicknell [1940]). This may harmonise with the statement that vitamin E will prevent the occurrence of experimentally induced muscular lesions but will not cure established disease. Hence, together with the work of Hjärre and Lileengen

(1936), some tentative doubt must also arise concerning the causal role of vitamin E deficiency in certain spontaneous muscular lesions in domestic animals.

A number of papers have appeared on so-called "stiff lamb disease" in the U.S.A. and on "white muscle disease" in young calves (see Vawter and Records [1937]), although the literature goes back long before this date. Cotchin (1948) recorded an outbreak in lambs in England and with literature other than that cited here; his description of the lesions tallies with the German accounts and with those on the American disease; he was unable to draw any succinct conclusion from a clinical therapeutic trial using various preparations of vitamin E. He also considered the similarity of the lamb lesions with those of experimental vitamin E deficiency, with those in paralytic myoglobinuria in horses, but seems to have overlooked Hjärre's paper. Jones and Reed (1948) give a model pathological record of a comparable case in a thoroughbred filly in which the muscular lesions were likened to the experimental dystrophy; they made an important additional suggestion that the muscles of dead foals and fœtuses should be examined after necropsy, and that the possible influence of vitamin E in the death of new-born foals was worthy of exploration.

No one could doubt the great resemblance in pathological type between the "weisses Fleisch," "stiff lamb disease," the lambs in England (Cotchin), and the foal of Jones and Reed, and all of these to the natural and experimental muscular lesions in laboratory animals (cf. Figs. 1 and 5). There is, however, a need for an unequivocal demonstration that in domestic animals vitamin E (tocopherols) is concerned ætiologically, and that this chemical substance has a curative effect, in naturally occurring myopathic conditions — in the same dramatic way that ascorbic acid has in scurvy or vitamin D in rickets of children. In clinical trials designed towards this end, because of the difficulty in assessing results, the use of muscle biopsy technique would appear to be essential.

For the purpose of this paper some allusion must be made to inflammatory conditions of skeletal muscle, excluding those due to viruses and bacteria which have some predilection to attack this tissue (e.g., in foot-and-mouth, infectious anæmia, anaerobic infections, actinomycosis, botriomycosis and phlegmonous conditions — an acute necrotising myositis has also been seen personally in experimental *Trypanosoma cruzi* infection of mice and dogs). Apart from descriptions of all these specific conditions, Nieberle and Cohrs (1949) elaborate on a peculiar muscular condition of cattle and pigs designated *Myositis chronica eosinophilica*. This has not been recorded in Britain or the U.S.A., and is characterised by diffuse, fibrous, thickened muscles (individual ones or groups). The lesions are notably unusual by the presence of marked inflammatory cellular infiltrations in which eosinophil leucocytes predominate; the same condition has been seen rarely in dogs (see Kuscher, 1940; Wirth, 1941; and Bloom, 1950). The cause is unknown, but beliefs that it is due to pre-existent sarcosporidial infestations have not been substantiated. Invasion of skeletal, and cardiac, muscle by these parasites is an extremely frequent observation in post-mortem examinations of all domestic, and most laboratory, animals but

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rarely of carnivora (*Sarcocystis bertami*, horse; *S. blanchardi*, cattle; *S. tenella*, sheep; *S. miescheriana*, pigs; and *S. horvathi*, fowls — according to Hutyra, Marek and Manninger). All pathologists who have seen these parasites in muscle (cf. Figs. 2a and 2b) no doubt have been impressed that they cause no immediate tissue reaction, and we do not know how long the Miescher's tubes, or psorosperm sacs, remain non-pathogenic (cf. *Trichina spiralis* invasion of muscle, Fig. 2c). They do not take long after birth to invade muscle, for I have seen them in very young lambs (cf. Fig. 2b). After death of the parasites inflammation may ensue around the parasites which may become calcified, and eosinophils may abound in the cellular infiltrations; to this lesion German authors (see Nieberle and Cohrs) have given the name *Myositis sarcosporidica*, but this is distinct from the *Myositis eosinophilica* in which parasites apparently are never found. There is, of course, a traditional belief that eosinophilic infiltrations signify parasitic infestation generally (also allergy), but there is no proof that parasites are concerned in this specific eosinophilic inflammation of muscle. It is doubtful if a purely lymphocytic myositis has been described in animals, but no great search of the literature has been made; prudence might be required in differentiating this from lymphoblastic or myeloblastic infiltration of skeletal muscles in the leucoses (leukæmia).

This introduction serves as a basis upon which some comments can be centred around the cases described. A consideration of these, and the papers cited, might be sufficient to indicate that there is some need for further critical work on muscular disorders of domestic animals.

Progressive Pseudo-hypertrophic Muscular Dystrophy (Dog)

Case 1.—Dog, retriever, M. 9 years. P.M. No. 128.46 (Registry of Vet. Path., Armed Forces Institute of Pathology Ac. No. 231285).

Clinical history

Slow progressive stiffness in hind limbs over period of six months; much worse in terminal stages of two months; on examination by the practitioner the hocks were almost on the ground (Fig. 3a), as if the *tendo Achillis* was ruptured, but this was normal; animal restless as if in pain; other general health good but animal obese; testes small and flabby; diffuse bilateral cataracts; timid disposition; thyroid and orchitic hormone given for three weeks; the treatment produced some reduction in weight but no improvement in limb condition; sores appeared on hocks; animal killed and autopsy done immediately.

Post-mortem examination

Anatomical findings. — Gross obesity; voluminous fatty deposits in subcutaneous tissues and other depots. No lesions in external genitals, mouth, ears; slight cataract both eyes. No lesions alimentary canal, liver, pancreas, gall-bladder. Kidneys normal in size, colour, shape and cortical pattern; bladder dilated with 300 c.c. clear urine; prostatic enlargement with nodular areas of adenomatous hyperplasia. Right testis contained a small 0.5 cm. interstitial-cell tumour; both testes small. Thyroid small for animal of this size. Some œdema

of lungs but no other lesions. Gross amount of pericardial fat; no abnormality of heart, valves, main vessels. Brain taken but not examined histologically. The *Mm. gluteus, biceps, semimembranosus, semintendinosus* and vasti group normal in colour and consistency. The *M. gastrocnemius*, both limbs, showed distinct pallid colour; further dissection of limbs made after fixation in formalin saline when muscles were designated. All muscles in hind limbs were normal apart from the *M. gastrocnemius*.

Histological findings.—Under low magnifications the contrast between an unaffected and affected muscle (*M. gastrocnemius*) was so striking as to be unmistakable. In both cross and longitudinal sections there is marked atrophy of whole fasciculi to the point of complete disappearance of many fibres and replacement with adipose tissue (Figs. 3b and 3c). The persisting muscle fibres show no signs of waxy degeneration, for striation is still apparent even in remnants of fibrillary structures; here and there are single fibres in which part of their structure is replaced by fatty vacuoles. There is no calcifiation and no parasitic infestation. Very few lymphocytes are scattered between the atrophied fibres and there are no pigment-laden histiocytes. Sarcolemmal proliferation is present in all areas of the sections wherever remnants of muscle are to be seen; along some fibres cut obliquely the nuclei are arranged in serried rows (Fig. 3c); in some places these form multinucleated masses. The vessels and peripheral nerves present in the sections are normal. Other incidental findings in this case include a small circumscribed nodule of interstitial-cell hyperplasia in one testis, a single macrofollicular cyst in the thyroid, and the adenomatous hyperplasia in some parts of the prostate. All pieces of tissue from other muscles of the hind limbs are normal.

COMMENT

The lesions in this animal are identical with those described for human cases of true progressive muscular dystrophy of the pseudo-hypertrophic type. They stand in contrast to the pathological changes in the other cases which follow, but resemble the few other descriptions of this muscular disorder—rare enough in man but apparently rarer still in animals. The data available does not permit of any statement being made on its hereditary nature. It is noteworthy, however, that the dog was adult (9 years) before symptoms appeared, was grossly obese, and from the history there might have been some testicular atrophy; cataract was also present. The treatment with thyroid and testicular hormone may have improved the atrophic changes in the testes, for the sections showed some degree of active spermatogenesis. It is possible that the condition might even have represented one comparable to those human cases with a Fröhlich-like syndrome; unfortunately, the pituitary was not examined. The bilateral affection of the *M. gastrocnemius* was quite unmistakable during life, and this alone would seem to be a disorder not hitherto recognised in the literature.

Neuropathic Muscular Atrophy (Sheep)

Case 2.—Sheep, cross-bred, F. Adult. P.M. No. 201.37 (Registry of Vet. Path., Armed Forces Institute of Pathology Ac. No. 231283).

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Clinical history

Symptoms of staggering, prostration and finally unconsciousness near lambing; diagnosis of pregnancy toxæmia made by practitioner and by workers in Institute of Animal Pathology, Cambridge University; recovery within a matter of days, but the sheep remained down and paralysed in all four limbs for eight weeks, when it was unfortunately shot in my absence; only pieces of muscle from the hind limbs were preserved for examination; all limb muscles were reported as being tough, fibrous and pale.

Histological findings

The outstanding feature is the dense fibrous tissue replacement of vanished and atrophied muscle; thick bands, and solid sheets, of collagenous fibres separate fasciculi or individual atrophied muscle fibres (Figs. 4a and 4b). In the collagenous matrix there are many arteries with distinctly hyperplastic walls; the small nerves present in the sections may be smaller than normal but show no other change with routine stains; the lymph vessels are dilated. The muscle fibres which persist are small on cross section, or thin and delicately tapering on oblique or longitudinal section; no difficulty is encountered in identification of transverse striation in H.E. preparations, but this is brought out better with iron hæmotoxylin. There is no infiltration by leucocytes, but there are a few lymphocytes and pigment-laden histiocytes; the pigment no doubt has been derived from myoglobin, for there is no hæmorrhage in the vicinity. There is little interfascicular fat and there are no sarcosporidia present. Multiplication of sarcolemma is mostly absent and never prominent.

Comment

There would appear to be little doubt that the paralysis of this ewe was a residual effect of some "toxic" damage done to the nervous system and which must have been most severe. The location of the damage and its nature are highly conjectural but from the little that is known about ovine neuropathology one might suggest that a lesion had involved the motor area, red nucleus or motor paths in the cord. It would be unwise to speculate further, but to note that the toxic effects of eclampsia on the human nervous system are well known. Whatever the precipitating cause of the paralysis is immaterial in some respects here; the muscles exhibit those lesions which are known to follow disruption of nervous control; these are simple atrophy and consequent fibrous tissue replacement, and this is classically demonstrated in this case.

There might be some dubiety about the original clinical diagnosis of pregnancy toxæmia, but the acuity of onset, general toxic condition leading to unconsciousness, and coma were all quite typical and the observations were made by competent observers. The only other disease of pregnant ewes with which confusion might arise is lambing sickness. In the report on sheep diseases by the National Veterinary Medical Association, Great Britain, and other literature (Hutyra, Marek and Manninger), no reference can be found indicating that there might be residual paralysis following either of these conditions. Further, it would appear, in spite of the fact that the signs and symptoms of

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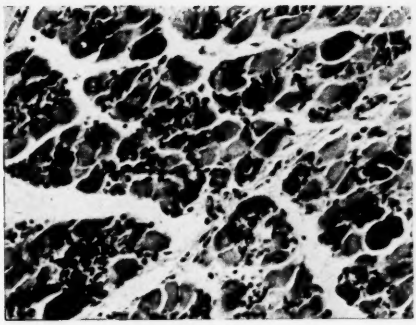

FIG. 1a

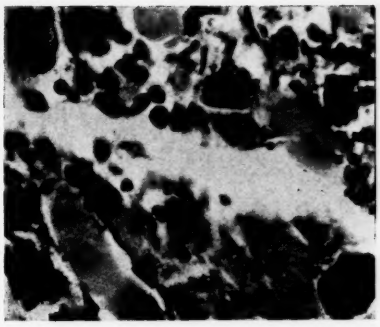

FIG. 1b

FIG. 1. *Muscular dystrophy*, spontaneous, rabbit (P.M. 326-46) from a number of cases observed in breeding stock; many muscles of body and diaphragm were speckled white. (a) Shows degenerative changes with swelling, fragmentation and loss of muscle fibres; granular calcification is frequent. H.E. x 100. (b) Same features under high magnification, showing type of cellular reaction and sarcolemmal proliferation. H.E. x 275 (AC231302).

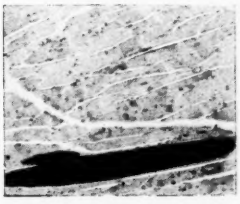

FIG. 2a

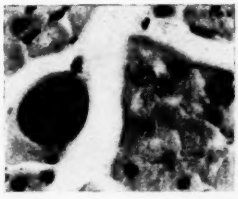

FIG. 2b

FIG. 2c

FIG. 2. *Sarcosporidial infestation of muscle*, (a) Cow, tongue (109-35); showing elongated Miescher tubes in muscle fibre with no reaction around. H.E. x about 60. (b) Heart, lamb, few weeks old. Showing isolated sarcocyst on transverse section with rim or normal muscle fibre around; no reaction. H.E. x 395. (c) Trichina spiralis (39-47) infestation of pig muscle; cellular infiltration around in which eosinophils are numerous. H.E. x about 70.

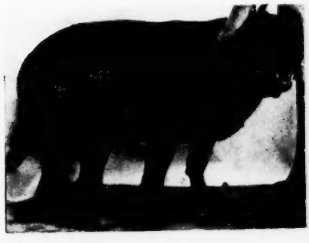

FIG. 3a

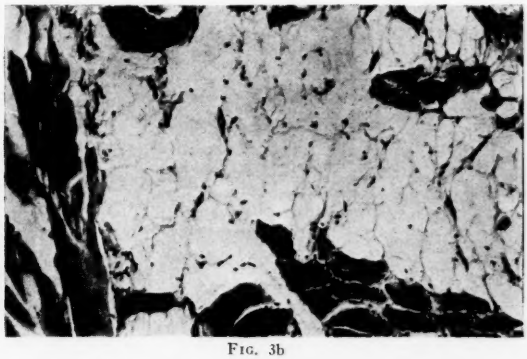

FIG. 3b

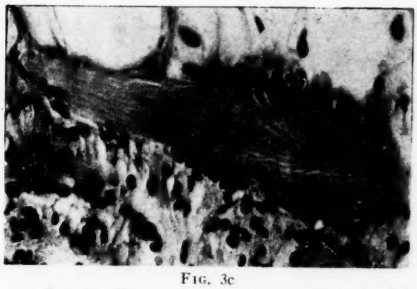

FIG. 3c

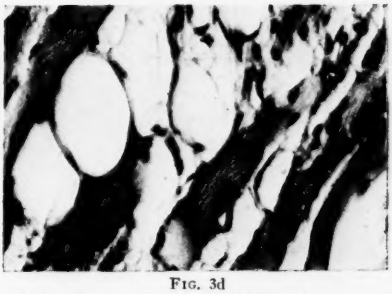

FIG. 3d

FIG. 3. *Progressive muscle dystrophy* (pseudohypertrophic). Case 1. Retriever. (a) Showing posture and obese appearance of dog just before death; hocks almost reach ground; (b) Showing marked loss and atrophy of muscle fibres with fat replacement; little fibrosis or cellular infiltration; sarcolemmal increase. H.E. x 75. (c and d). Same features at higher magnification. Clear persistence of striation. H.E. x 275.

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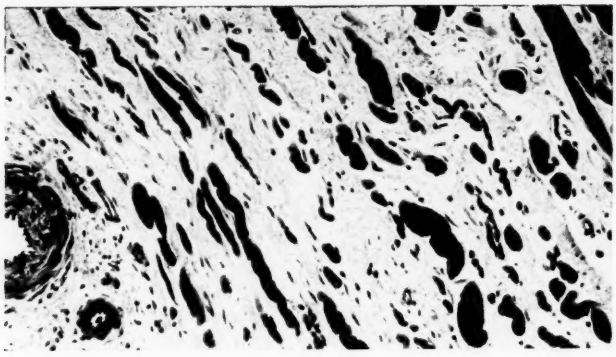

FIG. 4a

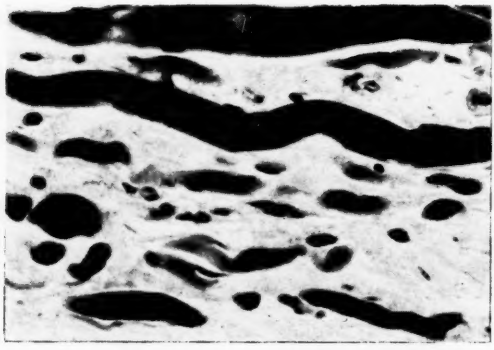

FIG. 4b

FIG. 4. *Neuropathic muscular atrophy*, Case 2, Sheep, residual effect of paralysis after pregnancy toxæmia. (a) Showing fibrous tissue replacement of vanished and atrophied muscle fibres; hyperplastic wall of artery; scattered lymphocytes and histiocytes. (b) Persistence of transverse striation in atrophied muscle fibres. H.E. x 275.

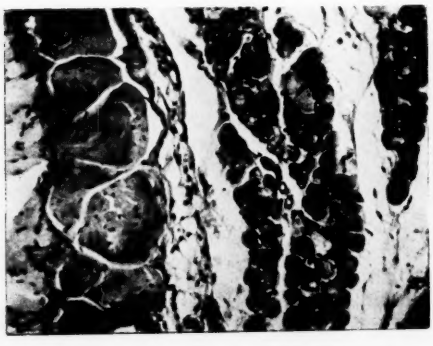

FIG. 5a

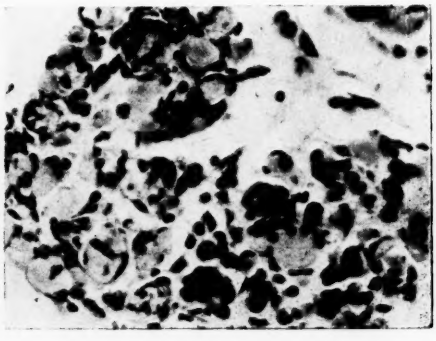

FIG. 5b

FIG. 5. *Muscular dystrophy* ("Weisses Fleisch"), Case 3, Lamb, few weeks old. Compare lesions with those in fig. 1 and 2 and with illustrations in books on nutritional muscular disorders in laboratory animals. (a) Showing isolated waxy type of degeneration of fibres; little inflammatory cell infiltration; no parasites present. Normal unaffected muscle at left. H.E. x 100. (b) Showing sarcolemmal proliferation, lymphocytic infiltration and little calcification. H.E. x 375.

(*Article by Innes, page* 131)

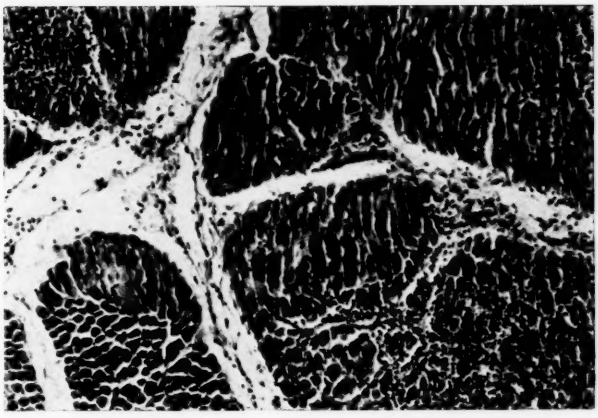

FIG. 6a

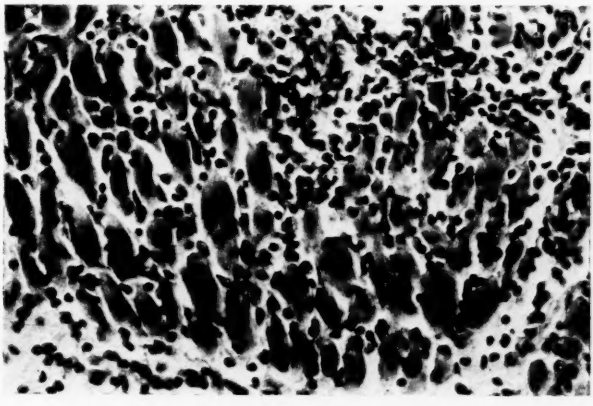

FIG. 6b

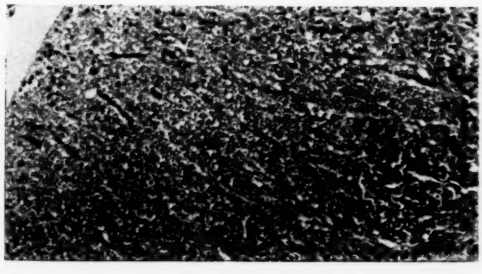

FIG. 6c

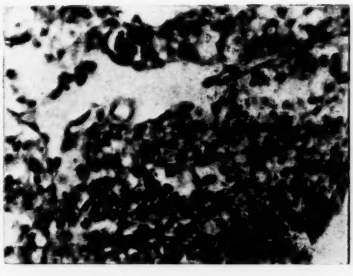

FIG. 6d

FIG. 6. *Lymphocytic histiocytic myositis, radiculitis, myelitis,* Case 4, Retriever. (a) Showing necrosis and atrophy of muscle fibres and crowded foci of lymphocytes and histiocytes; no waxy degeneration or calcification. H.E. x 100. (b) Higher magnification showing type of cellular infiltration. H.E. x 275. (c) *Spinal cord, lumbar region.* Showing focus of lymphocytic cellular infiltration in dorso-lateral column of white matter. H.E. x 75. (d) *Nerve root, spinal cord,* lumbar region. Showing loss of nerve fibres, some ballooning of persisting myelin sheaths, lymphocytic and microglial infiltration. H.E. x 275.

(Article by Innes, page 131)

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either of these disorders point to some nervous mechanism, I can find no reference to any pathological study of the nervous system ever having been made. This might give cause for reflection, for in the literature emphasis is entirely towards an explanation of the disease in terms of metabolic disturbance and blood chemistry.

Congenital Muscular Dystrophy ("weisses Fleisch") (Lamb)

Case 3.—Lamb, Welsh mountain. Few weeks old. P.M. No. 244.46 (Registry of Vet. Path., Armed Forces Institute of Pathology Ac. No. 231287).

Clinical history

One of a flock of eleven in which ewes were treated with copper to prevent swayback; this animal suspected of having swayback or cerebellar atrophy, and no doubt on casual examination these might have been possible diagnoses; lamb unable to rise, but when helped showed great inco-ordination and placed hind feet well forward under the body; in good condition; no opisthotonus; animal killed.

Post-mortem examination

Anatomical findings.—No lesions in any of the viscera, joints or nervous system—slicing of the brain being made after fixation. The *M. semitendinosus* in both hind limbs was extremely pallid (almost white) and flabby compared with normal red colour and firm consistency of adjacent muscles.

Microscopic findings.—There are no lesions in any part of the nervous system indicative of swayback or any other established nervous disease of sheep. A few Miescher tubes — sarcosporidia — are present in the cardiac muscle (Fig. 2b) without any tissue reaction around, but there are no parasites in any of the skeletal muscles examined. The muscle directly adjacent to the *M. semitendinosus* is normal. In affected portions of the latter some of the fibres are enlarged, waxy or hyaline, round in shape, and stain intensely with acid dyes, and this is all that is seen in some areas; the vessels and nerves are normal; there is almost no fat or inflammatory cellular infiltration. Directly adjacent to these same areas the fibres of some bundles have vanished, or persist as small atrophied ones, or they may be very thin and tapering and cross-striation is lost; some of the fibres are minute and spindle-shaped (Fig. 5). Lymphocytic and histiocytic infiltration is marked, but eosinophils are difficult to find. Sarcolemmal proliferation has been intense in places and constitutes a notable microscopic feature. Calcification is of a minor degree.

Comment

The features seen in this case appear identical with those described in "weisses Fleisch" of the German literature, in "stiff lamb disease," in white muscle disease of lambs in England (Cotchin), and in the dystrophy of the thoroughbred filly (Jones and Reed). The fact that only a single muscle was affected (naked eye) may not be unusual, for this is in accord with the findings of some workers that parts of one muscle, a single muscle, or the entire musculature may be affected in calves and sheep. Enough comment has been made,

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in the introduction, regarding the present position of ætiology of these peculiar muscular dystrophies which seem to occur in many animals. These are not true examples of progressive muscular dystrophy in the sense of the term used by neurologists. Some attention ought to be given to the work of Hjärre and Lileengen (1936) in Sweden on the possible causal role of vitamin C.

Lymphocytic/Histiocytic Myositis, Neuritis, Radiculitis and Myelitis (Dog)

Case 4.—Dog, retriever, M. 12 weeks. P.M. No. 100.46 (Registry of Vet. Path., Armed Forces Institute of Pathology Ac. No. 231284).

CLINICAL HISTORY

One of a litter of ten; bitch sired by own son; pup observed to be wobbly on hind legs about seven weeks; gait normal when moving in straight line but swayed if turning sharply; no pain on palpation at that time. Betaxan given daily for next two weeks. Animal became progressively worse, unable to walk upstairs, hopped with both hind limbs together; limbs became extended and stiff; in sitting position, legs extended straight under chest; no fever at any time; appetite normal, and little muscular wasting; animal killed and autopsy made immediately.

POST-MORTEM EXAMINATION

Anatomical findings. — No abnormalities in mouth, neck structures, alimentary canal, liver, pancreas, genito-urinary system, respiratory system, or cardiovascular apparatus. The hind limbs and lumbar region were dissected; all muscles appeared paler than normal and there seemed to be some disuse atrophy. The joints were normal, as were also the main peripheral nerve trunks.

Histological findings. — Sections were made from five separate portions of muscles of the hind limbs; all were affected in the same way. There are no wide areas of myonecrosis, remarkably little hæmorrhage, and there are no parasites. In less involved areas of skeletal muscle the lesions consist of small foci of lymphocytes and histiocytes separating small bundles of fibres; there are no polymorphonuclears or eosinophils, but a small number of plasma cells in these cellular collections. In more intensely affected parts this reaction is very marked (Figs. 6a and 6b), and these cells form foci as well as infiltrate between individual fibres and extend along interfascicular tissue; in these areas muscle fibres have undergone necrosis and atrophy. There are no pigment-laden histiocytes present. There is no waxy degeneration of fibres, almost no fibrosis, and sarcolemmal proliferation is of a very minor kind. This total picture seems to have affected all muscles of hind limbs and lumbar region quite indiscriminately. No inclusion bodies are found with the stains employed. A similar type of infiltration is present in short lengths of the peripheral nerves from both hind limbs; this is not so severe as in the muscles, and with the stains employed there appears to be no loss of myelin or changes in the axis cylinders. An identical reaction is also found in both the anterior and posterior nerve roots

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of all the sections of cord examined from the lumbar-sacral segments; in these roots there may be some ballooning and loss of myelin, but there appear to be no changes in the axis cylinders. Four sections of spinal cord were made from different levels; small cellular foci are present in the white matter of the ventro-lateral columns; there is no distinct loss of myelin, no hæmorrhage, and no meningeal infiltration; most of the motor neurones appear normal, but some of the anterior horn cells show chromatolysis resembling that seen in retrograde axonal change of other conditions. There are no inclusion bodies in ependymal cells, vascular endothelium, glia or neurones. Unfortunately, the brain and spinal cord above the lumbar region were not examined. There were no lesions in periosteum, bones or intervertebral discs of the lumbar vertebræ.

COMMENT

The history of the animal indicates that this disorder was subacute and lasted about a month before the animal was killed. During this period no fever was reported, but it is clear that later the condition was painful and disabling. The lesions described indicate that the disease embraced a disseminated lymphocytic/histiocytic myositis, neuritis, radiculitis and myelitis. It is possible that a more complete examination of the skeletal muscles might have revealed that the disease was more widespread than appeared. The combination of lesions in the nerve roots, peripheral nerves and cord raises issues which cannot now be answered, but the case might be unique in many respects. No similar cases of myositis have been reported in the literature, but then the question might be raised as to how often skeletal muscles are examined histologically in the course of routine post-mortem examinations. The nervous lesions raise the problem as to whether the process was part of an encephalitic syndrome, and no answer can be given. No inclusion bodies were found and there were no other features, at least in the spinal cord, characteristic of most varieties of the canine encephalitides. The lesions were most severe in the muscle: hence one might consider that the process had been an ascending one. There appears to be no resemblance between the myositis and that seen in the Coxsackie virus infection, yet the possibility of some virus infection being responsible should be considered. In experimental Coxsackie virus infection of mice (see Melnick [1950]), the disease is acute and mostly fatal; the lesions consist of hyaline necrosis, œdematous separation of muscle fibres, loss of cross-striations, and mononuclear infiltrations. It is unknown whether this virus (originally recovered from human patients suffering from diseases simulating poliomyelitis, summer grippe, and epidemic myalgia) is pathogenic for dogs. The combination of lesions in muscle, nerve roots and cord might also remind one in some respects of the rare Landry-Guillain-Barré syndrome of man (see Haymaker and Kernohan [1949]). It is manifest that skeletal muscle might be worthy of examination in the canine encephalitides.

General Comment

Diseases of the muscular system in animals could be studied further with advantage to animal pathology in general. From my collection of material five

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distinct types of lesions are described and illustrated, and some comparison is made with known conditions in man and experimental animals. This may stimulate thought by clinicians that some of the conditions are not so uncommon as this record indicates. Pathological examination of the tissues affected revealed the true nature of the disorders, which at least emphasises pathology is essential in helping to elucidate what may appear to be a clinical puzzle.

There are other lessons to be learnt. I have drawn attention before to the fact that no diseases are recognised until they are found by looking with great care. One need only scan the veterinary literature of the last twenty years for this to be confirmed, and a long list of conditions could be compiled which might have been discovered years previously if more meticulous methods of pathological examination had been used. Veterinary pathology has still a long road to travel before it achieves the profundity of human pathology, and this is not said in any deprecatory sense of past achievements. There are many reasons why this is so, but lack of volume of material studied and inattention to the lessons to be learnt from medical work have played a part. In veterinary work, unfortunately, also the question of economics always intrudes, and in the end we are constantly influenced by a consideration of what is, or is not, important.

In conducting autopsy examinations of animals there is usually an immense amount of bias in selection of tissues for histological work; in the case of skeletal muscles (from my own experience) no doubt they are mostly ignored unless macroscopical changes are very obvious. Perhaps this article may help to focus attention on the pathology of muscular disorders.

Summary

(1) A review of the literature on the comparative pathology of myopathies is given.

(2) Cases of (a) spontaneous muscular dystrophy in a rabbit; (b) true progressive muscular dystrophy (pseudo-hypertrophic type) in a dog; (c) neuropathic atrophy (d) " weisses Fleisch " (lamb); and (e) lymphocytic/histiocytic myositis, neuritis, radiculitis and myelitis (dog) are all described and illustrated.

(3) In domesticated animals the causal relationship of vitamin E to spontaneous muscular dystrophies is still not elucidated.

(4) There appears to be an analogy with *Myotonia congenita* (Thomsen's disease) of man in the disorder of goats described in the U.S.A.; there are no recorded diseases parallel to *Dystrophia myotonica*, *Myasthenia gravis*, and amyotrophic lateral sclerosis of man.

(5) The occurrence of a lymphocytic/histiocytic myositis and radiculitis in a dog might suggest the possibility of the existence of a hitherto unrecognised micro-organismal agent with a predilection for attack on canine muscle; we do not know to what extent canine neurotropic viruses attack peripheral nerves or muscle.

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Acknowledgments

My thanks are due to Mr. J. Berger, M.R.C.V.S., Alderley Edge, for providing me with the clinical histories of the two canine cases. I am also deeply indebted to Col. T. C. Jones, Registry of Veterinary Pathology, Armed Forces Institute of Pathology, Washington, D.C., for his co-operation in making available facilities for preparation of the microphotographs.

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THE EXMOOR PONY AND THE PLACE OF OUR HORSES IN RURAL ECONOMY

By J. G. SPEED, M.R.C.V.S., and MISS M. G. ETHERINGTON,
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In contemplating the decline of the horse, we have been encouraged to survey its rise.

Far from accepting present conditions, we feel that in the College where William Dick countered prejudice and brought light and reasoning into the veterinary art, it is a proper duty to preserve the heritage which will be an invaluable asset in the future.

Broadly speaking, Britain is rapidly being divided into separate economies, rural and urban. At present the urban economy is dominant, and to a great extent controls the nation's policy. This has been brought about by forces which were inevitable, but equally inevitably a return to a balanced economy will happen.

In the rural economy each item must be given the full consideration due to the separate individuals of a population. It is easy enough in a short-term policy to flatter the one most immediately useful while flouting the one whose uses momentarily may be overlooked. Economic factors, and most of all its constitutional deterioration, have combined to bring it about that the horse is for the time being in eclipse. In the smaller nations these facts are quickly being recognised, since there is less detail to obscure the economic picture.

The veterinary profession has a responsibility in the readjustment of the nation's economy and will be lothe to be shown remiss or time-serving when the emergency arises.

For a start, we have tried to appreciate to what the faults and weaknesses of our modern horses may be traced, and inevitably such research has led us to a study of the whole history of the horse in Britain, and the ramifications of this study have extended, if somewhat tentatively, to Europe, Asia, and Africa. It may be stated that, were it not for disease and sickness, a place for the horse in agriculture and transport would be unassailable; and could we find a robust, reliable animal with native health, and which could winter outside and maintain its vigour, the horse could be at once reinstated. This last comment is provoked by the knowledge that horses have survived and thrived in Britain in an ecological succession which sought no help from man, whilst now that man's influence has become dominant in the affairs of the horse, its doom has appeared imminent.

In looking for a horse which will fit into the new economy we have examined the factors which could have reduced the constitutional health of our horse population in the past, and two were outstanding. One of these is its husbandry, and it is hoped to work experimentally on that problem; the other is selective breeding, and already we have seen that therein lies much of the explanation of an approaching tragedy. This tragedy can be made a blessing if by reducing

PLATE I

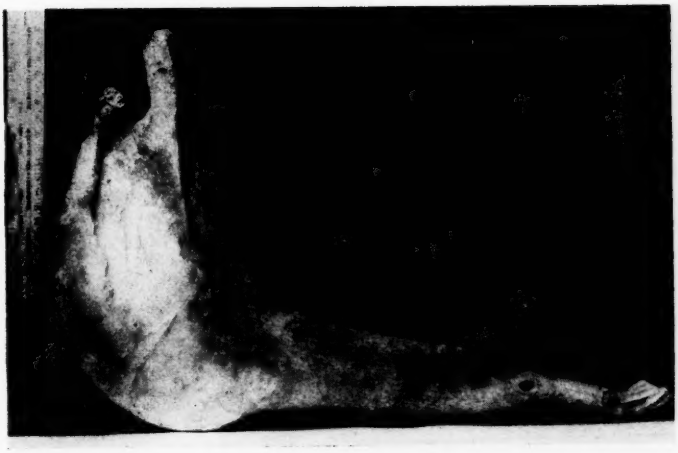

FIG. 1
An Arab type mandible, A.D. 1937.

FIG. 2
A mandible from an Arab type horse in Britain, 250 B.C.

(*Article by Speed and Etherington, page* 144)

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FIG. 3
Five-year-old Exmoor mare.

FIG. 4
The Celtic horse of 250 B.C.

(*Article by Speed and Etherington, page* 144)

FIG. 3
Mandible of Prjevalsky's wild horse.

(*Article by Speed and Etherington, page* 141)

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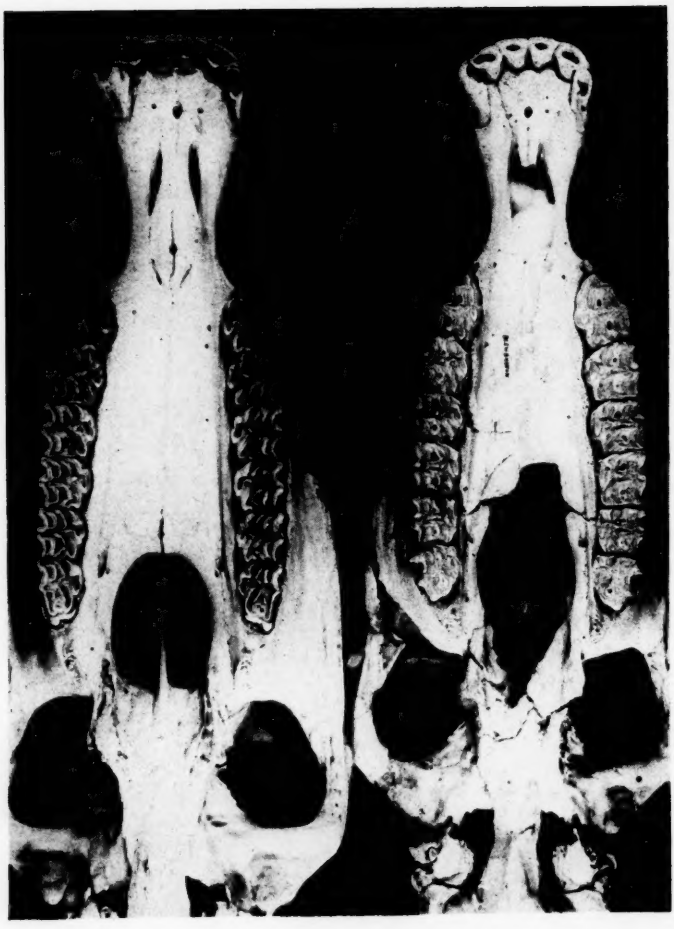

FIG. 6
A selective breeding experiment of 250 B.C., repeated in A.D. 1950, showing a similar dental anomaly of the right corner incisor. The skull on the right is from the recently-discovered Blewburton A-B Iron Age Celtic horse.

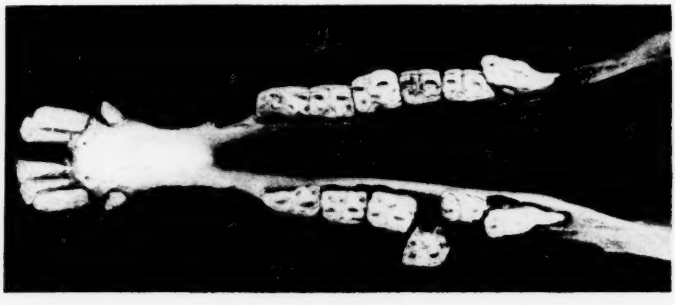

FIG. 7
Showing the narrowness of the mandible and set of the incisors of the imported sire.

(Article by Speed and Etherington, page 144)

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the numbers of the horse it prunes back to the stock from which vigorous new growth can be got.

Anatomical examination and dissections of some of our modern breeds of horses have shown unexpected defects of conformation which in themselves would ultimately ring the death knell, and an added misfortune is that the show-ring and its consequent emphasis on a sire's name will frequently laud these very defects. In this context we have so far paid special attention to the dentition, and whilst it is a truism that a horse must earn his living with his teeth, it is also a truism that the teeth must have jaws capable of carrying them. Distortion of the facial bones by selective breeding may result in what passes for a neat little muzzle to please the eye, and the owner of this muzzle may survive for quite a long spell of life if carefully hand-fed, but an examination of such a mouth after the death of the animal can only arouse pity and dismay in the minds of those who intend that the horse shall survive and be ready to take its proper place in our whole economy.

Such selective breeding has been carried on, judging by the fossil evidence, for at least three thousand years in Britain, and the woeful evidence of its lack of success has been preserved by the careful inhumation of the prized animals.

With this knowledge, and with the dire straits in which we find our horse population to-day, it seems that the time is more than ripe for a close study and research into these problems.

We selected for our first study the Exmoor pony, since it is a breed of horse which shows less of the defects of human interference in its economy than any other British breed. Of late years even the Exmoor has suffered from the attention of the breeders to the dictates of popular and romantic fancy as to what a horse should look like. To the prejudiced eye of an ecologist or an anatomist, teeth set all askew in a degenerate small head, and a coat which tends to be silky and sun-repellent rather than protective, and a tail set high, on a rump which reaches to the heavens rather than down to work, may seem to be defects; but the breeders, being practical men, know that it is the popular fancy that pays the price: some few breeders have adhered to the traditional breeding policy—or rather, in the case of the Exmoor pony, have left it to the salutary care of herd laws and to the rigid selective influence of a rugged environment—and so it is still possible to find a small and diminishing number of animals which can be truly called British horses.

The dwindling number of these horses is a matter for concern, but with such specimens as we have managed to secure we have made a search for their ancestry and lineage and have been surprised and greatly encouraged by their significance. It is a matter for more than mere curiosity to find that the ancestors of the Exmoor pony lived in or near their present habitat before the advent of man; that their likeness appears on the murals, friezes and plaques of ancient Syria, and Iraq; that in European Neolithic cave paintings they have an honoured place; whilst in the Gobi, until the gun of the hunter recently exterminated them, their cousins, Prjevalsky's wild horse, roamed free on the steppes.

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This is an interesting lineage, but it is more to the point to appreciate that the robust constitution and the tractability of this, our own wild horse, have been applied in the past to produce the foundations of many of our once famous breeds. It is unknown or unrecorded just how much the native horse has influenced these breeds, possibly because its special colour factors are recessive, and because anything native is taken for granted and not prized until it is lost; but the occasional experiment carried out to-day more than confirms all the legend of the past and an anatomical examination enhances this appreciation.

A letter from Gunnar Bjarnason, the scientific adviser to the Ministry of Agriculture in Iceland, has also given us much encouragement since he, in the simpler economy of his own country, can see more clearly the fundamentals of the case for the horse at home and abroad, and he has already set out upon the first stages of restoring the horse to its proper place. His difficulties are in some ways greater than our own, because in the smaller environment of Iceland the possibility of a pure breed surviving in isolation are remote, and so he has the problem of the mixed, imported, and "selected" breeds to deal with.

The Basuto pony, too, has been "improved" so much by the use of foreign sires that it is becoming unfit for its environment, and an attempt is now being made to revive the native stock with its splendid traditions.

Using our pure native foundation stock, we intend to show by controlled experiments the processes by which our breeds formerly reached their perfection.

Fortunately for themselves and for Britain, the Exmoor pony requires no housing, hand-feeding, or sick nursing, and so has survived—to the present at least—as the nucleus of a foundation stock which can be used to bring about the rise of the horse in Britain.

It may be to the point to show photographed examples of some of the defects and deformities which can be produced by faults in selective breeding.

Acknowledgments

We are grateful to the British Museum (Natural History) for permission to use their specimens 1924.5.4.1 and 1945.6.11.1; to the Reading Museum for the Blewburton material; and to Mr. R. S. Hood and Mr. E. Roberts, of the Royal (Dick) Veterinary College, for their care and help with the photography and technical work respectively.

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A STUDY ON THE BLOOD SUPPLY OF THE CAMEL'S HEAD

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Introduction

A STUDY on the blood supply of the camel's head is of interest to both veterinary science and comparative anatomy.

Lesbre (1906) states that the carotid artery and the jugular vein are concealed by the transverse processes of the cervical vertebræ; the thyrolaryngeal artery is voluminous; the occipital A. resembles that of equines; the internal carotid is very small, and the external carotid is not distinct from the internal maxillary, and extends to the superior dental canal. It gives the following branches:—

1.—The facial, given at the inferior extremity of the parotid gland; this, in turn, detaches (A) the superior labial A., which passes on the surface of the masseter M. and then under cover of the zygomatic and lachrymal muscles to reach the nostrils; (B) the inferior labial A., which runs on the lateral surface of the mandible; and (C & D) the anterior and posterior auricular arteries.

2.—The lingual A., which gives off the pharyngeal at its origin.

3.—The inferior dental A.

4.—The posterior deep temporal A.

5.—The anterior deep temporal A.

6.—The artery to the rete mirabile of the cranial cavity.

7.—The sphenopalatine A.

8.—The small palatine A.

He reports that the ophthalmic A. forms an orbital rete mirabile. He admits the presence of an internal jugular vein.

The object of this present work is to study fully the blood supply of the camel's head.

Material and Method

Material for this work was obtained from Cairo slaughterhouse. Ten heads with the whole of the necks attached were used. They were from mature male and female Sudanese camels. The skin was intact. The specimens were injected, via the common carotid artery, with 10 per cent formalin solution, to which some colouring material was added. Some pure calcium sulphate was added to harden the injected mass and prevent staining of the surrounding structures.

The Blood Supply of the Camel's Head

The blood supply of the camel's head is derived from the common carotid artery and its branches.

The dissection of the vessels was accomplished with some difficulty, due to the small space available and the general anatomical form of the skull.

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Many vessels were buried deeply in narrow grooves, whilst vessels which in other domesticated animals take origin from points distant from each other are represented in the camel as arising from good-sized single trunks. Generally, the vessels are smaller in calibre, and the veins are provided with a greater number of valves than is the case in other domesticated animals.

The Common Carotid Artery

This artery, during its course in the cervical region, lies on the ventral aspect of the neck, concealed in a deep groove formed by the prolongations of the transverse processes of the cervical vertebræ in a ventral direction in conjunction with the multifidis cervicis M. and the ventral group of muscles of the neck. The artery is well protected, concealment being necessary for an animal possessing such a long and exposed neck. For bleeding purposes, the artery can be exposed easily by a ventral incision between the sixth and seventh cervical vertebræ. This is the usual point for severing the vessel in the slaughterhouse.

On approaching a level ventral to the caudal extremity of the axis, the artery appears from the before-mentioned groove, crosses the trachea and the larynx (the œsophagus also on the left side) obliquely to gain a dorsal position directly under the caudal extremity of the atlas ventral to the rectus capitis ventralis major. On the lateral surface of the great cornu of the hyoid bone, the artery clearly divides into two terminal branches—the external carotid and the internal maxillary arteries. This is contrary to the statement of Lesbre that the division is indistinct. Here the artery is related laterally, in succession, to the omohyoid M. (separating it, as usual, from the jugular vein), the suprapharyngeal lymph gland (separating it from the occipital vein and the mandibular salivary gland) and the stylohyoideus M. (separating it from the digastricus M. and the occipitomandibularis M.).

Numerous collarteral branches are detached from the artery before its bifurcation :—

1. *The thyrolaryngeal* is detached from the ventral aspect of the parent trunk at the superior extremity of the thyroid gland, forming a right angle with the carotid.

2. *The occipital A.* Although of considerable size, this is a collateral branch from the common carotid. It is detached from the dorsomedial aspect of the parent trunk, coursing upward and forward medial to the suprapharyngeal lymph gland and mandibular salivary gland (the latter gland in the camel is situated between the parotid gland and the wing of the atlas). In the alar canal of the atlas, the artery divides, as usual, into two branches—the anterior or cerebro-spinal and the posterior, recurrent or descending. The latter passes through a bony canal, beginning from the alar canal and opening on the dorsocaudal part of the wing of the atlas. This canal represents the foramen transversearium of equines.

Besides muscular and glandular branches (mandibular), the occipital A. gives off a large branch in the fossa atlantis which runs upwards and forwards

PLATE I

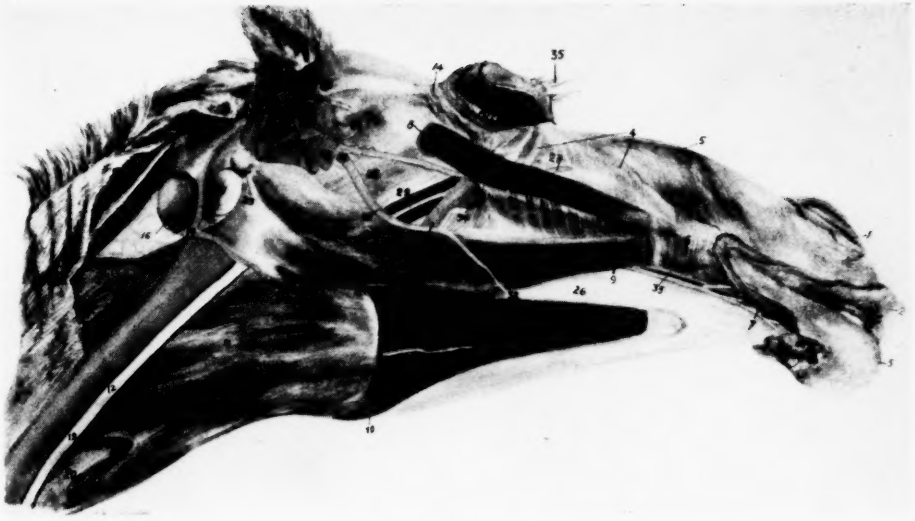

FIG. I

4, Malaris M.; 5, Nasolabialis M.; 6, Buccinator M.; 7, Orbicularis oris M.; 8, Zygomaticus; 9, Resorius M.; 10, Mylohyoid M.; 11, Omohyoid M.; 12, Jugular vein; 17, Ventral branch of the second cervical nerve; 19, Sternomastoid M. (Sternocephalicus); 21, Mandibular salivary gland; 22, Parotid salivary gland; 27, Dorsal buccal nerve; 28, Masseter M.; 29, Parotid duct; 30, Facial artery; 31, Facial vein; 32, Superficial branch of the dorsal buccal nerve; 33, Ventral buccal nerve and inferior labial artery and vein; 34, Veins from the buccal venous plexus; 35, Thick bush-like long hair for protecting the eye from the sun glare.

FIG. II

11, Tendon of nasolabialis M.; 12, Poll glands; 13, Branch from the occipital artery to the pool glands; 15, Frontal; 16, Frontal vein passing through foramen leading to the osseous canal mentioned in the text; 17, Angularis oculi vein; 18, Cutaneous vein joining the angular vein; 19, Mediodorsal branch of the malar A.; 30, Branch from the lachrymal A.; 21, Branches from the dorsal branch of the zygomatic nerve; 22, Complexus M.

(*Article by Tayeb, page* 147)

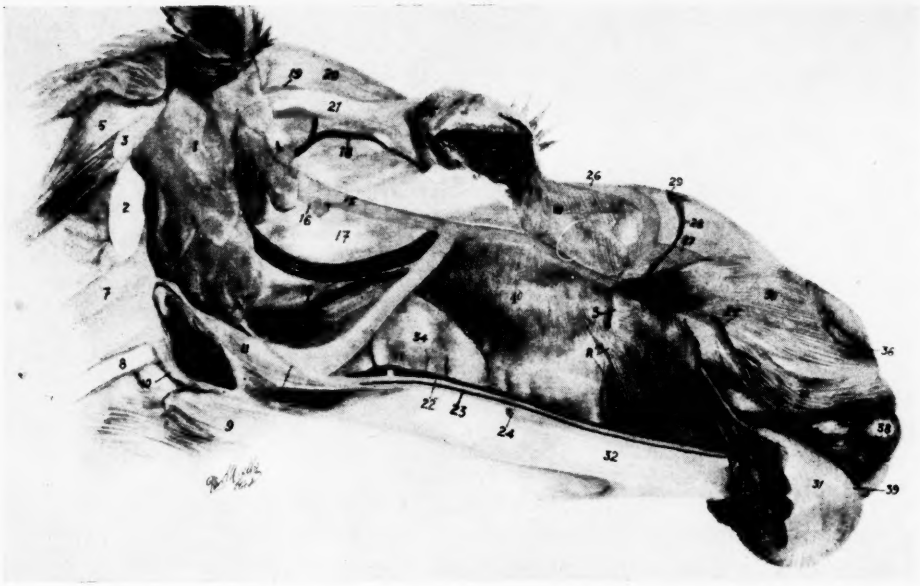

FIG. III

1, Parotid gland; 2, Mandibular salivary gland; 6, Mandibular lymph gland; 7, Jugular vein; 8, Sternomastoid M.; 9, Omohyoid M.; 10, Lingual vein; 11, Common facial vein; 12, Ventral buccal nerve; 13, Facial artery; 14, Parotid duct; 15, Dorsal buccal nerve; 17, Masseter M.; 18, Transverse facial artery; 19, Anterior auricular A.; 22, Inferior labial vein; 25, Superior labial A. with the satellite vein; 26, Angularis oculi vein; 27, Dorsal nasal vein; 28, Branch from the infra-orbital A. compensating partly for the angularis oculi A.; 30, Caninus M.; 34, The ventral large buccal gland.

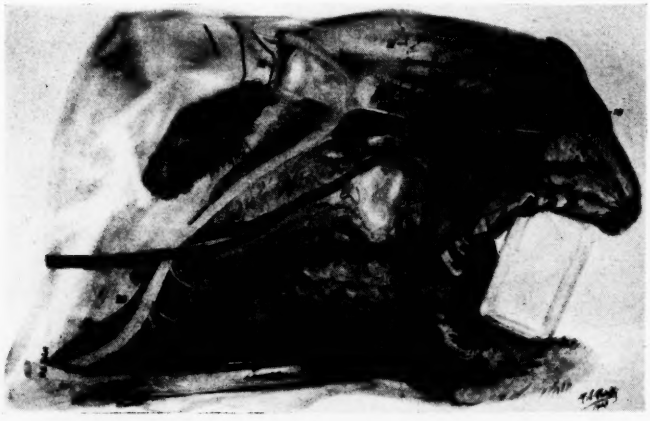

FIG. IV

1, Facial A.; 2, Facial V.; 7, Infra-orbital A.; 8, Dorsal nasal V.; 11, The collateral branch from the infra-orbital; 13, The angularis oculi V.; 14, Superior labial A.; 16, Dorsal angularis oris A.; 17, Labial branch of the superior labial A.; —, Nasal branch of the sup. labial A.; 20, Ventral branch of the dorsal terminal branch of the facial; 25, The ventral terminal branch of the facial artery in company with the satellite vein and the continuation of the buccinator nerve; 27, The inferior labial artery and vein and the ventral buccal nerve; 30, Massetric branch from the facial A.; 31, Superior labial V.

(*Article by Tayeb, page* 147)

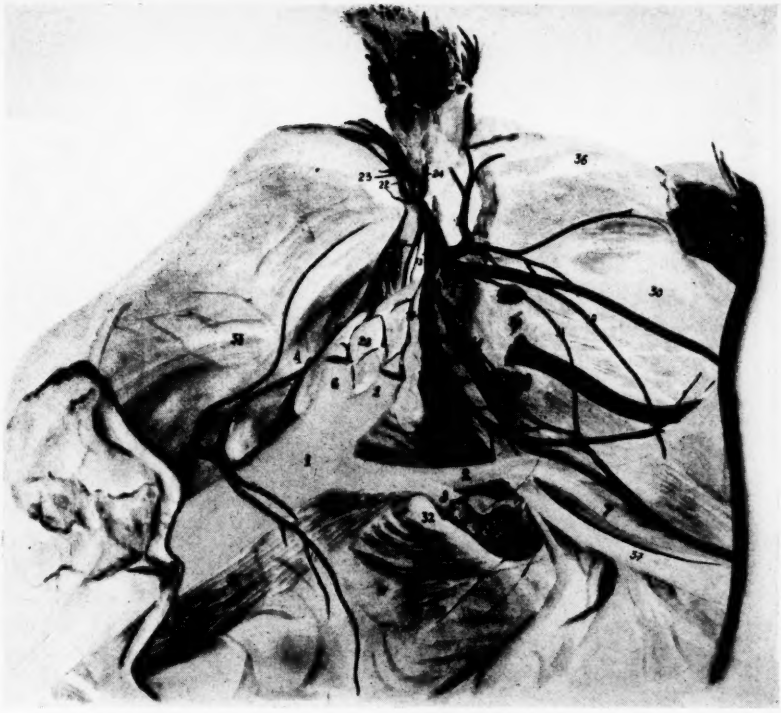

FIG. V

1, Jugular vein; 2, Facial vein; 3, Lingual V. (the vein here joining the facial instead of joining the jugular directly which is a variation); 6, Stump of great auricular vein; 7, Inferior labial vein; 17, The external carotid terminating into 18, Superficial temporal and 19, External maxillary or facial; 20, Anterior auricular A. in company with auriculopalpebral nerve; 21, Transverse facial A.; 22, Posterior branch of the posterior auricular A. dividing into 3-4 branches; 24, The lateral branch of the posterior auricular A.; 25, Inferior labial A.; 26, Branches to the parotid gland; 38, Branches from the submental A.

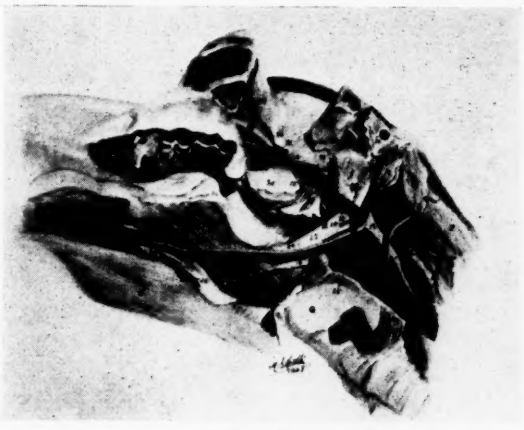

FIG. VI

1, Common carotid A.; 2, Anterior laryngeal A.; 3, The dorsal branch from its usual origin; 4, Occipital A.; 5, Internal maxillary A.; 6, External carotid A.; 7, Auriculomeningeal A.; 8, Posterior branch of the posterior auricular A.; 9, Lateral branch of the same; 10, Posterior meningeal A. coming from below the stylomastoid foramen; 13, Occipital vein; 17, The ventral branch to mandibular salivary gland; 18, Lingual artery; 19, Submental A.; 20, Sublingual A.; 22, Pharyngeal A.; 23, Great cornu of the hyoid; 27, The common trunk detached from the internal max. A. giving the middle meningeal A. (28) and the tympanic A. which is concealed—the rest of the trunk can be seen in Fig. VII; 29, Branch from the middle meningeal, the levator palati M., ophthalmic M.; 34, Lachrymal A.; 38, Ventral cerebral vein.

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PLATE IV

FIG. VII

1, Common carotid A.; 2, Internal maxillary A.; 3, External carotid A.; 4, Auriculomeningeal artery; 5, The common trunk from the int. max. A. giving the inferior alveolar A. (6) and the single deep temporal A. (7); 8, A variation in the origin of the trans. facial A.; 9, Buccinator A.; 10, Lingual A.; 12, Sublingual A.; 18, Thyrolaryngeal A.; 19, Variation of the origin of the dorsal muscular branch from the common carotid; F., Facial vein with its root A. angularis oculi and N. dorsal nasal veins.

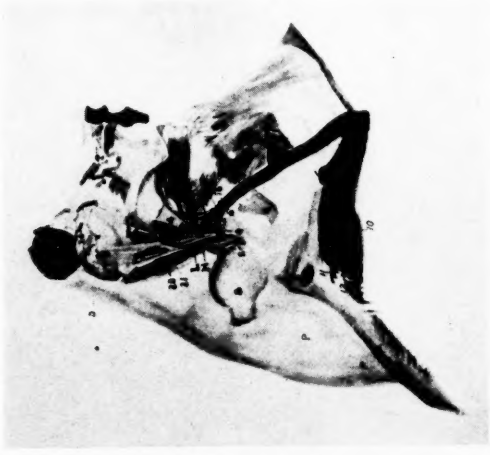

FIG. VIII

SS., Anterior meningeal arteries derived from the medial aspect of the sphenopalatine A.; 7, Internal maxillary A.; C., Common carotid; 8, External carotid A.; 8', External carotid stump; 9, Auriculomeningeal A.; 10, Lateral branch of the posterior auricular A.; 11, Posterior branch of the same; 12, The posterior meningeal concealed in the deep groove; 16 and 18, Stumps of the internal max. A.; 15, Stump of the ophthalmic A.; 16', Stump of the buccinator A.; 14, The common trunk detached from the int. max. A.; 17, The common trunk for both palatine arteries; 19, Part of the lachrymal A.; 20, Supraorbital A.

(Article by Tayeb, page 147)

in front of the parent artery, to reach the skin in the region of the poll, where it supplies the poll glands in male subjects.

Contrary to the statement of Lesbre, the condyloid A. (prevertebral) and posterior meningeal (mastoid) are not detached from the occipital artery in the camel.

3. *The anterior laryngeal A.* is a small branch given from the ventral aspect of the carotid opposite the origin of the occipital; it gives branches to the pharynx.

4. *The internal carotid* is a collateral branch from the common carotid. Contrary to the statement of Lesbre, it is of good size, taking origin from the dorsomedial aspect of the parent trunk, and shares in the formation of the rete mirabile of the cranium.

5. *The condyloid A.* arises from the parent trunk just in front of the origin of the internal carotid. It accompanies the hypoglossal nerve to the hypoglossal foramen.

6. *The lingual A.* is the largest collateral branch of the common carotid; arising from the ventral aspect of the parent trunk under cover of the stylohyoideus M. It follows the caudal borders of both the great and middle cornu of the hyoid bone. The hypoglossal nerve is in contact with the lateral aspect of the vessel, only separated from it by the hyoglossus M.

Beside muscular branches, the lingual detaches two important collateral branches:—

(a) The pharyngeal or ascending palatine arises from the anterior aspect of the lingual just at its origin from the common carotid. It passes on the medial aspect of the great cornu to accompany the pharyngeal branch of the glossopharyngeal nerve.

(b) The submental A. takes origin from the lingual just at the junction of the great cornu with the middle one. It runs on the posterior aspect of the lingual for some distance.

7. *Two arteries* to the mandibular salivary gland are detached from the common carotid; the ventral is the smaller taking origin behind the lingual and passing to the apex of the gland, after detaching a branch to the supra-pharyngeal lymph gland. The dorsal one passes to the anterior border of the gland above the exit of the mandibular duct.

8. *A large muscular branch* usually takes origin from the parent trunk just behind the occipital: it passes dorsally over the rectus capitis ventralis major and the obliquus capitis inferior. In some specimens it arises opposite the origin of the thyrolaryngeal A.

The Terminal Branches of the Common Carotid

As mentioned before the artery divides on the lateral surface of the great cornu into two terminal branches—the dorsal or external carotid and the ventral or internal maxillary arteries.

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The External Carotid.

The artery courses upward and slightly forwards on the lateral surface of the great cornu; on approaching the tendon of origin of the stylohyoideus M. it turns sharply laterally to its original direction to appear beneath the deep face of the parotid gland in the space bounded posteriorly by the digastricus and anteriorly by the angular process of the mandibular ramus where it divides into two terminal branches :—

(1) The superficial temporal A. resembles the same vessel in equines; and (2) the external maxillary or the common facial A., which is described later. Each of these branches is at right angle to the parent trunk.

The only collateral branch given by the external carotid is a large branch termed *The Auriculo-meningeal A.* The artery is detached at the tendon of origin of the stylohyoideus M. and continues the original direction of the parent trunk. It passes upwards in a deep groove formed by the lateral surface of the great cornu medially, the paramastoid process of the occipital dorsally and the mastoid process of the petrous ventrally to divide after a short distance into two terminal branches—the posterior auricular and the posterior meningeal arteries. Both arteries course in the same direction as their parent trunk.

(a) *The posterior auricular artery* is the more superficial of the two branches; after a short distance it divides into lateral and posterior branches as is the case in equines.

(b) *The posterior meningeal artery.* Just below the stylomastoid foramen, it turns sharply in a postero-medial direction, curving round the base of the ear, to gain a deep oval depression on the caudal aspect of the nuchal crest where it passes through a foramen (absent in both equines and bovines) leading to the parieto-temporal canal.

The External Maxillary of the Facial Artery

This is the longer of the two terminal branches of the external carotid, it courses ventrally following the posterior border of the mandibular ramus beneath the deep face of the parotid gland crossing the medial aspect of the jugular vein and the ventral buccal branch of the facial nerve successively on its way through this region. In the neighbourhood of the angle of the jaw, it passes on the surface of the masseter M. and then under cover of the parotid gland and the facial vein. Beneath the vein it changes its direction, going forwards and upwards to appear in a space bounded by the parotid duct dorsally and the facial vein ventrally. Here the artery is felt easily, and this is a good position for feeling the pulse, the vessel being covered only by the skin and a very thin layer of the cutaneous faciei M.

At the anterior border of the masseter M., the artery ends by dividing into two branches.

1. *The ventral branch or middle labial artery.*—By its size, the artery appears to be the continuation of the parent trunk coursing downwards and then forwards, forming a small arch beneath the facial vein, to run on the dorsal surface of an extremely large ventral buccal gland in company with the con-

tinuation of the buccinator nerve and a satellite vein. On its way to the lower lip, it detaches a branch to the angle of the mouth—the ventral angularis oris artery; it gives also many branches to the buccal glands which form a thick continuous layer on the lateral side of the mouth cavity. Although owing to the presence of an inferior and a superior labial arteries, appearing to be a middle labial, yet it represents the inferior labial of equines.

2. *The dorsal branch.*—This is the smaller. It runs dorsally under cover of the parotid duct and the dorsal buccal nerve ending in two branches. The ventral is the larger, turning downwards beneath the nerve and the duct, then, after coursing forwards on the surface of the buccal glands and giving some branches to them, it disappears about 3 cm. caudal to the angle of the mouth. The dorsal one is expended in the zygomaticus M. and the cutaneous faciei M. in this region.

It is evident from this description that the facial artery does not reach the nostrils, which is contrary to the statement of Lesbre.

Beside several small branches to the parotid gland and a single branch to the mandibular lymph gland, the facial detaches three important collateral branches:—

(a) A large massetric branch is detached at the angle of the jaw.

(b) A long but slender inferior labial artery is detached at the angle of the jaw and runs forwards on the lateral surface of the mandible in company with the inferior labial vein and ventral buccal nerve.

(c) A smaller massetric branch is detached just before the bifurcation of the parent trunk.

The Internal Maxillary Artery

By its size and direction, it constitutes the continuation of the common carotid artery. It courses upwards and forwards from the lateral side of the great cornu of the hyoid to the orbital cavity after passing through an arch formed by the pterygoid process which represents the alar canal in equines, then continues its course through the infra-orbital canal as the infra-orbital artery. For convenience of description, the artery may be described in three stages.

First Stage.—From the lateral surface of the great cornu to the arch mentioned above. In this stage the artery is related medially to the great cornu and the vertical part of the palatine bone; bridging the space between the two, it lies in a thick layer of fat. Laterally, it is related to the pterygoideas medialis and lateralis muscles respectively.

Several collateral branches are detached from it in this stage.

1. *The sublingual A.* arises from its ventral aspect on the lateral side of the cornu. Its course resembles that of the same artery in the horse.
2. A small branch to the pterygoideas medialis arises just in front of the preceding one.
3. A small branch to the levator veli palatini takes origin from the medial side in front of No. 2.

4. *The common trunk.*—This is the largest and most important collateral branch in this stage. It passes upwards and outwards between the pterygoid muscles and then downwards to rest on the lateral surface of the pterygoid lateralis forming an arch, the convexity of which is directed dorsally. On the lateral pterygoid M. the artery divides into two terminal branches—the inferior alveolar (inf. dental) which resembles the same vessel in the horse, and the second, the deep temporal artery.

The deep temporal A.—In calibre this artery is equal to the inferior alveolar artery and represents both the anterior and the posterior deep temporal arteries in equines. Contrary to the statement of Lesbre, the anterior deep temporal A. is absent in the camel.

The common trunk also gives the middle meningeal and the tympanic arteries as collateral branches.

Second Stage or orbital division of the int. max. A.

This extends from the arch to the entrance of the infra-orbital canal. Medially, the artery rests on the bone, then passes over the lateral surface of the orbital tendon of the pterygoid medialis M. Laterally, it is covered by the pterygoid lateralis M.

Its collateral branches are as follows:—

1. *The buccinator A.* arises from the dorso-lateral aspect of the parent trunk in the alar arch. It resembles the same artery in the horse.
2. *The ophthalmic A.* is also detached from dorsal aspect of the parent trunk in the alar arch, close to the origin of the preceding A. It is smaller in size than that of the horse. Its branches are: (a) the lachrymal. This is larger and longer than that of the horse. After giving a branch to the lachrymal gland, it proceeds to supply the region posterior to the supra-orbital process. (b) The supra-orbital. This is small and sends a branch to the frontal sinus. The ethmoid A. is not detached from the ophthalmic. No rete mirabile is formed by the ophthalmic artery. This is contrary to the statement of Lesbre.
3. *The sphenopalatine A.*—The artery is of considerable size. Arising from the ventral aspect of the parent trunk, it passes forwards on the lateral side of the orbital tendon of the pterygoideas medialis M. in company with the sphenopalatine nerve. From the medial aspect of the artery, several branches are detached parallel and in close contact with each other. Each forms an arch, the convexity of which faces anteriorly. They pass backwards through the F. rotundum ventral to the cranial nerves in this region to join in the formation of the rete mirabile of the cranium.
4. *The great and the lesser palatine arteries* are detached by a common trunk from the lateral aspect of the parent vessel.
5. *The ethmoid A.* arises from the internal maxillary opposite the origin of the common palatine trunk.

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Third Stage through the infra-orbital canal.

The upper part of the infra-orbital canal in the camel is formed of a deep groove separated from the floor of the orbital cavity by hard fibrous tissue which also forms the roof of this groove converting it into a canal.

On traversing this groove, the artery detaches the malar artery. The malar A. divides immediately into two branches. The lateral branch appears on the surface of the malar bone below the orbicularis oculi, giving branches to it and the malaris M. The medial one passes dorso-medially over the lachrymal sac, giving a branch to the latter, and then appears on the surface of the frontal bone dorso-caudal to the medial canthus of the eye. This artery partly compensates for the absence of the angularis oculi A. in the camel.

The Infra-orbital Artery

In addition to its importance as the continuation of the internal maxillary artery, this constitutes the main blood supply to the upper lip, nostrils and the lateral nasal region. After its emergence from the infra-orbital foramen, the artery ends in two terminal branches:—

1. *The superior labial A.* is the ventral and larger branch. It passes forwards and slightly downwards under cover of the caninus M. and in company with the ventral (labial) branch of the infra-orbital nerve. On the anterior surface of the upper lip, it divides into a labial branch to the upper lip and a nasal branch which runs dorsally to anastomose with the dorsal nasal.

In addition to the small branches given on either side, the artery gives a collateral branch which runs ventrally to the angle of the mouth—the angularis oris dorsalis.

2. *The lateral nasal artery* is the dorsal and smaller branch. After coursing a short distance it divides into two branches. The ventral runs forwards representing the lateral nasal A. of equines, passing ventral to the lateral wing of the nares to anastomose with the nasal branch of the superior labial. The dorsal branch passes dorsally to the lateral wing of the nares representing the dorsal nasal of the horse.

The infra-orbital artery detaches the following collateral branches:—

(a) *The superior dental A.* is detached from the medial aspect of parent trunk. It passes through the superior dental canal in company with the superior alveolar nerve. It is a long and slender artery.

(b) *The second branch* is detached from the infra-orbital just before its bifurcation. It penetrates both the caninus and nasolabialis muscles to appear on the surface of the latter, partly compensating for the absence of the angularis oculi artery.

The following variations were observed in two specimens.

In the first on the left side, the transverse facial artery was detached from the common trunk and (see the int. max. artery) coursed backwards in company with the superficial temporal nerve. It passed through the space between the angular process and the mandibular ramus and then proceeded as usual.

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The same variation was also seen in the second specimen, but in this case it was on the right side.

The Veins of the Head

The Jugular Vein

It begins behind the temporo-mandibular articulation by the union of the superficial and internal maxillary veins.

The superficial temporal V. is formed only by the transverse facial and the anterior auricular veins.

The internal maxillary is the backward continuation of a single trunk resulting from the union of the vena reflexa and the buccinator veins. Its tributaries are the same as those in the horse, with the addition of the ventral cerebral vein.

Unlike the case in other domesticated animals, the jugular vein passes beneath the deep surface of the parotid gland without penetrating its substance. Caudal to the angle of the jaw the jugular receives its three most important tributaries simultaneously, after which the vein increases in size, suddenly reaching about 3-4 cm. in calibre. Then it courses very much obliquely ventro-caudally on the surface of the omohyoid M. and in contact with the dorsal border of the sternomastoid muscle (sternocephalicus). Here the vein is accessible for intravenous injections or venesection.

The tributaries of the jugular in the region of the head are as follows:—

1. *The posterior auricular V.* is much larger than the same vein in the horse. In addition, it receives tributaries from the region of the poll glands in the male. It traverses the parotid gland ventro-medially to emerge beneath the lower third of its deep surface where it joins the jugular.

2. *The massetric* resembles that of the horse and joins the jugular above the facial.

3. *The external maxillary or facial V.*—The importance of this vein lies in its being (a) superficial in most parts of its course, (b) the longest and largest of the tributaries in the region of the head, and (c) the surgical and medical importance of the angularis oculi V., one of its roots. The latter forms a venous inosculation on the surface of the frontal and nasal bone through its direct communication with the frontal vein forming a large valveless trunk. The two trunks of both sides are joined by a large, wide and valveless communicating vein.

The frontal vein joins the ophthalmic by passing through an osseous canal opening on the medial wall of the orbit. The ophthalmic vein communicates with the temporal vein before its entrance to the F. rotundum on its way to the cranial cavity.

On the lateral surface of the maxilla close to the nasal bone, the facial V. is formed by the union of the angularis occuli and the dorsal nasal vein (the lateral nasal usually joins the facial at the same point). The facial vein has a large size; it inclines backwards on the lateral surface of the maxilla and malar

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bones, successively under cover of the malaris and zygomatic muscles, following both the anterior and ventral borders of the masseter and forming a conspicuous curve. Caudal to the angle of the jaw, it joins the jugular under cover of the resorius M., and is related to the inferior extremity of the parotid gland. It crosses the deep face of the dorsal buccal nerve under the zygomatic M., but the parotid and the facial artery are crossed laterally by the vein.

The vena reflexa receives a large trunk, the tributaries of which are the satellites of the palatine and sphenopalatine arteries. The trunk joins the vena reflexa before the formation of the internal max. V.

The lingual V. joins the jugular below the facial.

The occipital receives the dorsal cerebral vein medial to the mandibular salivary gland. The jugular receives the occipital at the inferior extremity of the mandibular salivary gland at a level with the facial and lingual veins.

Contrary to the statement of Lesbre, the deep or internal jugular vein is absent.

REFERENCE

Lesbre, M. F. X.: "Recherches Anatomiques Sur Les Cameldes." Extract: Archives du Museum, His. Nat., Lyon, 1906.

XUM

STUDIES ON THE BLOOD OF DOGS

V.—Hæmatological Findings in Dogs Suffering from (a) Teeth Diseases; (b) Skin Diseases

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(a) Teeth Diseases

INSPECTION of the teeth of the dogs studied in this research was carefully made at their entry to the School Hospital and during post-mortem examination. Many of the dogs which were otherwise normal in appearance were affected with teeth diseases, the most important of which were *pyorrhœa alveolaris* (or suppurative pyorrhœa) and caries. The former, a septic inflammation of the gums, in which suppuration is produced, is a common condition in aged dogs. Pus was noticed to exude from the alveoli and the breath of the animal acquired a somewhat characteristic foul odour of a most persistent nature. The gums were dark red, swollen and bled easily. The latter affection, i.e. caries, which is the disintegration of the discoloured dental tissues, is soon followed by cavity formation within the tooth. The tooth pulp becomes exposed and liable to be infected. As long as the opening through the enamel is free, pus will be discharged without further complication; if it becomes closed with food particles, etc., an abscess develops at the root and the pus burrows out into the adjacent parts.

Results and Observations

Blood samples from 20 dogs were taken to determine the hæmatological picture in diseases of the teeth.

Red Blood Corpuscles

The average count per c.mm. was 5,172,000, which is slightly lower than normal. The range was between 3,410,000 and 6,300,000 per c.mm.

Hæmoglobin Percentage

The average percentage was 56, which is also slightly lower than the average percentage of the normal dog. The range was between 37 and 75 per cent.

Colour Index

The index was relatively higher than the normal index, averaging about 0.54 and ranging between 0.46 and 0.69.

Reticulocytes

The count was within normal range; it varied from 0 up to 5 per cent, with an average of 1.3 per cent.

Total Leucocyte Count

Marked leucocytosis was recorded in these cases. The leucocyte average was 15,030 per c.mm., the range being from 4,200 to 23,200 cell per c.mm.

XUM

Differential Leucocyte Count

Relative and absolute neutrophilia was present. The neutrophiles amounted to 70 per cent, ranging between 37 and 85 per cent of the total leucocytes.

Eosinophiles had a normal percentage, averaging 4.6 per cent and ranging between 0.3 and 15.2 per cent of the total leucocytes.

Basophiles were also within normal range, amounting to 0.05 per cent.

Lymphocytes were reduced to 15.3 per cent, with a wide range varying from 3 to 46 per cent of the total leucocytes.

Monocytes showed no significant change in the range or average. They varied from 4 to 26 per cent, averaging 10 per cent.

Schilling Index

Myelocytes were absent from the peripheral blood. Juveniles and stabs were increased to an average of 0.4 and 15.8 per cent, respectively. The stab forms reached a maximum of 41 per cent of the total leucocytes. The segmented cells were also increased. The range was from 28 to 72 per cent, with an average of 53.9 per cent of the total leucocytes.

Discussion

The above observations agree well with those stated by Pepper and Farley (1934), who wrote the following: "Under pyorrhœa alveolaris, just as with other types of focal infection, various claims of characteristic blood changes have been made. It has frequently been said that focal infection induces a lymphocytosis in the circulating blood; this is without doubt incorrect. Unusually no change is found in the blood. Occasionally a slight or moderate secondary anæmia may seem to be related to long-standing oral sepsis as if the process is severe and associated with definite gingivitis, a slight neutrophile leucocytosis may appear with slight shift to the left in the neutrophile picture."

C. E. De Camp (1936) states that dogs with chronic nephritis, suppurative pyorrhœa and chronic endometritis show low hæmoglobin values.

Summary

The blood of 20 dogs in which diseases of the teeth were detected was examined.

A slight secondary anæmia characterised by a low red cell count, low hæmoglobin percentage and a high colour index was present. There was an increase in the total leucocyte count with an absolute and relative increase in the number of neutrophiles.

This type of leucocytosis is found chiefly in conditions in which there is an inflammatory process produced by pyogenic organisms. There was a lymphopenia with no significant change in either eosinophiles, basophiles or monocytes.

There was a slight shift to the left in the neutrophiles.

XUM

(b) Skin Diseases

Nineteen dogs with skin affections came under notice. About half the cases, namely 10, were diagnosed as eczema. The others were mange and ringworm affections; in these the parasite and its identity was determined by careful microscopic examination.

Accordingly, the cases were classified into two main groups:

(a) Non-parasitic or eczematous cases.

(b) Parasitic, including mange and ringworm.

Results and Observations

Red Blood Corpuscles

There was no significant change from the normal average in the two groups. For the first group, i.e. dogs affected with eczema, the range was between 3,600,000 and 7,610,000 per c.mm., with an average of 5,498,000 per c.mm. For the second group, i.e. dogs affected with mange and ringworm, the range was between 4,200,000 and 8,600,000 per c.mm., with an average of 5,360,000 per c.mm.

Hæmoglobin Percentage

This also was within the normal range; it varied from 50 to 70 per cent, with an average of 57 per cent, for the first group, and from 50 to 62 per cent, with an average of 59 per cent (slightly higher than normal) for the second group.

Colour Index

This was slightly higher than normal. The calculated limits were between 39 and 64 for the first group and between 19 and 66 for the second group, the average being 53 and 56 for the two groups, respectively.

Reticulocytes

These ranged from 0.8 to 2.2 per cent, with an average of 1.3 per cent (within normal figures), for the first group, and from 0.2 to 2 per cent, with an average of 0.8 per cent (slightly lower than normal), for the second group.

Total Leucocyte Count

There was a slight leucocytosis in the first group and a marked leucocytosis in the second group. The range for the former was from 9,400 to 18,400 per c.mm., with an average of 12,820, and, for the latter, from 10,800 to 20,000 with an average of 14,220 cells per c.mm.

Differential Leucocyte Count

Neutrophile cells were within normal limits. The range was from 62 to 74 per cent and from 50 to 76.2 per cent of the total leucocytes for the two groups, respectively. The averages were 66 and 60.7 per cent for the total leucocytes for the first and second groups.

There was a marked increase in the eosinophiles above the normal. The range for the first group was from 3.3 to 13.5 per cent, and for the second group from 1.1 to 21 per cent of the total leucocytes, the average being 7.7 and 7.5 per cent, respectively.

Basophiles were very slightly increased. The averages were 0.08 and 0.4 per cent for the two groups.

Lymphocytes were slightly decreased in the two groups. The range was for the first group between 3 and 22 per cent, and for the second group between 5 to 30.3 per cent of the total leucocytes, the average being 12 and 17 per cent of the total leucocytes.

Monocytes were markedly increased in the first group and slightly increased in the second group. The range for the first group was between 8 and 21 per cent, and for the second group between 13 and 23 per cent of the total count, the average being 14.3 and 17 per cent of the total count.

The Schilling index was within normal limits.

Discussion

The blood picture of the skin diseases was, in all respects, within normal limits if we except a leucocytosis accompanied by eosinophilia, monocytosis and lymphopenia, which was slight in the non-parasitic but well-marked in the parasitic (mange and ringworm) groups.

It is reported by different investigators that in various skin affections the eosinophiles are increased in the peripheral blood. Welch (1909-1910) reports finding eosinophilia amounting to 5 to 11 per cent in eight severe cases of follicular mange in the dog and that in four light cases he found no increase. He also found an increase in the same variety of cells in six cases of eczema in the same species. He states that the increase depended more upon the intensity of the dermatitis, which is manifested by pruritis and irritation, than upon the nature and extent of the disease.

Pepper and Farley (1934) state that the eosinophiles may reach about 5 to 15 per cent in scabies and occasionally higher in herpes zoster, but the increase is less marked in psoriasis, urticaria and eczema. Eczema and urticaria and the associated eosinophilia may both be manifestations of an allergic reaction to a foreign protein. Their statements agree well with our findings in reporting that there is rarely any significant change in the hæmoglobin percentage or red cell count. Interest has centred chiefly on the differential count of the white cells and particularly on the question of eosinophilia. The total white cell count is frequently increased, but more often as a result of an actual eosinophilia than from any neutrophile leucocytes.

Scabies was found by Schamberg (1912), and later by Hayman and Fay (1921), often to have an associated eosinophilia. The latter authors found

5 per cent or more of eosinophiles in 32 of 55 cases of scabies, the maximum figure being 15 per cent.

Authorities differ widely concerning the occurrence of eosinophilia in eczema. This is readily understood when one remembers the varying causes responsible for the different conditions grouped under this term. It is probable that when an allergic basis for the eczema exists, and this applies with equal force to urticaria, some degree of eosinophilia will be found. This explains the findings of as high as 45 per cent in one patient with eczema and none in another. Basophiles are said occasionally to be increased in the blood of patients with skin conditions.

Kracke and Garver (1937) state that eosinophilia is said to be a constant finding in a number of skin diseases, including psoriasis, pemphigus, pruritis and the types of eczema that are allergic in nature. The presence of eosinophilia is so remarkably constant in the allergic states that this finding in various skin conditions would indicate that the disease in question, in all probability, rests on an allergic basis.

According to Osgood (1931) the degree of eosinophilia which occurs with skin lesions depends more on the severity of the lesion than on the nature of the disease. It is specially marked in cases which are characterised by the formation of large blebs (pemphigus) and exfoliated dermatitis.

Magner (1938) noted that it occurred in eczema (usually slight) and it may be well marked in scabies and in herpes zoster.

Blount (1930-31) reports that eosinophilia is observed in certain skin diseases (where the greater the area of skin involved the higher the percentage of eosinophiles).

Price-Jones (1935) stated that skin diseases in the human subject are often due to mixed infection, but the coccal element is dominant. Leucocytosis is usually only slight. Relative counts of lymphoid and leucoid cells may be marked by the presence of eosinophilia. Often the blood may present only very slight variations from the normal. Increase in relative and absolute numbers of eosinophiles is sometimes met with in skin affections.

Whitby and Britton (1939) state that any infective or irritative conditions of the skin may give rise to an eosinophilia. The phenomenon is found fairly constantly in pemphigus, dermatitis, scabies, psoriasis and eczema. The amount of eosinophilia appears to bear some direct relation to the intensity of the infection and the extent of the affected skin.

Todd and Sanford (1937) state that eosinophilia has been observed in a large number of skin diseases. It probably depends less upon the variety of the disease than upon its extent.

Summary

The blood picture of 10 dogs affected with eczema and that of nine dogs affected with either mange or ringworm showed no significant change from

the normal except a moderate leucocytosis in the first group and a marked leucocytosis in the second. Eosinophilia, monocytes and lymphopenia were predominant features of these affections.

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REVIEWS

KOEGEL, A. Nutztierparasitologie für Tierärzte, Landwirte und Nutztierhalter. Band I. Protozoologie und Entomologie unter Mitarbeit von Hermann Bellow. (Parasitology of useful animals for Veterinary Surgeons, Farmers and Keepers. Volume I. Protozoology and Entomology, with the collaboration of Hermann Bollow.) 9½ x 6¼ in., vii + 339 pp., 130 figs., 103 refs. Stuttgart, F. Enke Verlag. 1950. Price: 30 DM unbound; 32.70 DM bound.

THIS book is the first of two volumes and deals with Protozoology and Entomology; the second volume will deal with Helminthology. The general principles of Parasitology are referred to in the first 24 pages, then follows the Protozoology section comprising 102 pages, and finally the Entomology section comprising 200 pages. A list of references and an adequate index complete the volume. After an introductory part, which includes the general morphology and biology of unicellular organisms, the section on Protozoology is arranged systematically and provides information on the classification, morphology, development, hosts, pathogenic importance and treatment of numerous species of protozoa, with which are included spirochaetes and organisms of doubtful systematic position, such as sarcosporidia. The section on Entomology is arranged on much the same basis but also provides information on the importance of arthropods as vectors of disease and the control of species or groups. Mention is also made of insects that are predocious.

Although this book appears to refer mainly to conditions in Germany, tropical parasites and parasitic diseases are also included. The host animals considered are the usual farm livestock, including poultry, fur-bearing animals, fish, bees and silkworms; several parasitic diseases of humans are also described.

The sub-title of this book informs the reader that it is intended for veterinary surgeons, farmers and livestock breeders. All these persons may derive benefit by reading its pages but it seems to be written mainly for the veterinary surgeon to aid him in diagnosing and treating parasitic infections. It is debatable if the subject matter, carefully arranged from the systematic point of view and using the latest nomenclature, will be completely comprehensible to the farmer and breeder since systematics demand specialised zoological knowledge. Nor are they likely to be interested, for example, in the 60-odd species of Culicines and 20-odd species of *Simulium* and their geographical distribution.

The author will doubtless take the opportunity of correcting some errors when a second edition is contemplated; for example, *Allobophora* for *Allolobophora* on p. 163; *Wucheria* for *Wuchereria* on p. 263, and Rotschild for Rothschild on p. 322. Such minor defects are, however, offset by the excellence of the illustrations, the clear printing and the good quality of the paper and binding, at least in the case of the bound volume which the reviewer had before him.

HORSE AND RIDER: EIGHT CENTURIES OF EQUESTRIAN PAINTINGS. Introduction by Geoffrey Grigson. London: Thames & Hudson. Price 12s. 6d.

THIS book is a collection of 64 plates in photogravure and two plates in colour depicting horses and their riders over many centuries. We see these through the eyes of great artists, such as Rubens, Van Dyck, Rembrandt, Velazquez, Goya and our own Ben Marshall and George Stubbs. Some of these artists, such as Gericault, Marshall and Stubbs, painted real horses; others evidently did not see horses with an ordinary man's eye, but with true artist's licence they depicted their subjects so that they resemble allegorical beasts. There are fat, fiery, prancing abnormalities which seem to be able to stand on one leg or on the air, and such as we see on the "roundabouts" at a fair or scattered in monumental form about the streets of London. No doubt the colouring and technical skill displayed in the originals entitle them to fame in posterity, and in some way compensate for the atrocious anatomical contortions or distortions.

In spite of these criticisms, the book will be of interest to all who love and admire the most gallant and beautiful of all the domestic beasts.

Geoffrey Grigson introduces one to the pictures, and at the same time he gives an interesting survey of the evolution of the riding horse.

A study of the harness of both man and animal is in itself a study in evolution.

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THE DOG-LOVER'S WEEK-END BOOK, by Eric Parker and A. Croxton Smith. Illustrations by Mcl. Brown. London: Seeley Service Co., Ltd. Price 12s. 6d.

THERE are a few people to whom a dog makes no appeal. They miss much of life, and, above all, the blessing of "A heart to love you till you die."

Now, most folks like dogs. They are intrigued by their intelligence, pleased by their flattery, for dogs are the greatest flatterers ever, amazed at their energy and ability, and also, and not least, they gain pleasure from the contemplation of canine grace and form.

The man who talks or writes of dogs is always assured of an appreciative audience. The two authors are gifted with their pens, but they have also been at pains to cull from others and over a wide field. The result is a book full of story and anecdote and much factual information. There are accounts of dogs in prehistoric Britain, in legend and history, police dogs, war dogs, and dogs in sport and work.

There is information on kennel club functions, on shows, on breeds, management and feeding. All this is intermixed freely with stories of sagacious dogs, cunning dogs and faithful dogs.

Towards the end there is a chapter titled, "Veterinary." The best part of this alludes to "Pepys' Diary."

Finally, there is a glossary, and in this one can find a fair interpretation of many of the words and phrases used by the fancy.

I read this book with great interest and pleasure. It is a book you can "make a meal of," or you can pick it up, open it haphazard, and you will find immediately something to hold your attention.

The authors are to be congratulated on their choice of artist.

ABSTRACT OF CURRENT LITERATURE

EN HURTIG-FARVNINGSMETODE TIL ADSKILLELSE AF LEVENDE OG DODE SPERMIER VED HJAELP AF EOSIN-NIGROSIN. (A Rapid Staining Method for Differentiation between Live and Dead Spermatozoa with Eosin-Nigrosin.)* By Erik Blom.

In this short communication the author has described a simple, rapid but improved method for differentiation of the live and dead spermatozoa.

The following is the technique:—

(1) Prepare two solutions: (a) 5% bluish eosin, and (b) 10% nigrosin, both with distilled water.

(2) Place on the slide a drop of fresh semen and stir it very carefully with a glass rod for a few seconds with solution (a) using twice the volume of semen.

* *Nord. Vet.-Med.*, 2: 58-61, 1950.

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(3) A drop (twice as big as the drop of the solution [a]) of the solution (b) is now added at the step No. 2 and the mixture stirred till it forms a thin smear.

(4) Dry the slide over a flame and examine under the microscope.

Results.—Two different kinds of spermatozoa are seen clearly on a brownish-violet background: living appeared as chromophobe whilst the dead spermatozoa are red.

J. K. GAN.

Publishers' Notices

THE BRITISH VETERINARY JOURNAL, with which is incorporated THE VETERINARY JOURNAL, is published monthly, and copy for advertisements should be in the hands of the publishers—Baillière, Tindall and Cox—not later than the 20th of the preceding month, if proof is required. Tel.: Temple Bar 3386.

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